Grass tetany in a herd of beef cows

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Abstract — Five cows in a herd of 15 cattle that had just been turned out onto lush pasture after having over-wintered on poor quality hay died suddenly. Biochemical profiles collected from the cadavers revealed reduced serum levels of magnesium, urea, and beta-hydroxybutycate. Classical grass tetany (hypomagnesemia) was diagnosed on postmortem examination.

Résumé — Tétanie d'herbage dans un troupeau de vaches de boucherie. Cinq vaches d'un troupeau de 15 qui avaient été mises dans un pâturage luxuriant après un long hivernage avec un foin de mauvaise qualité sont mortes subitement. Les profils biochimiques effectués à partir des cadavres ont révélé des niveaux sériques réduits de magnésium, d'urée et de bêta-hydroxybutyrate. Une tétanie d'herbage classique (hypomagnésémie) a été diagnostiquée à l'examen post-mortem.

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Grass tetany (hypomagnesemia) occurred in a beef cattle herd in May 2003 in Ontario. The 15 crossbred cattle had been over-wintered on poor quality hay and then turned out onto a "new," rapidly growing spring grass pasture 2 wk prior. These cattle had not been supplemented with any additional food sources, such as roughage, trace mineral, or supplement concentrate, at that time.

The presenting complaint on May 13th (day 1) was 2 cows found dead: 1 death had occurred the previous week, the 2nd on the morning of day 1. The latter was found under a tree after a thunderstorm, but no evidence of lightning strike was noted after examination of the cadaver. A necropsy was performed within 24 to 48 h of death, and no abnormal findings were noted. However, assessment of the field revealed a pond, as well as a pile of burned garbage that included paint cans and pieces of plastic and metal. The differential diagnoses included lead toxicity, blue-green algae toxicosis, and toxicity of unknown origin. A 1-mL blood sample was collected from the jugular vein of the most recently found cadaver, placed in an ethylenediaminetetraacetic acid (EDTA) vacutainer, refrigerated, and then submitted for toxicology at the Animal Health Laboratory (AHL) (University of Guelph, Guelph, Ontario). The serum lead level was determined to be within normal limits. On day 8, 2 more cows were found dead in the morning, and a third cow was reported to have charged a farmhand, collapsed, convulsed, and then died. This series of events caused the owner to request a field visit by the veterinarian. A necropsy examination was performed within 30 min of death. The gross postmortem examination revealed no abnormal findings, aside from poor body condition. Samples of kidney, lung, liver, brain, spleen, and rumen contents were collected into 10% buffered formalin, as well as being packaged and frozen for histopathologic and lead toxicologic, respectively. Rumen content was also placed in a sealed plastic bag, frozen, and sent to AHL to be held pending histopathologic results. The lead levels in the kidney were within normal limits. Histopathologic examination revealed multifocal perivascular hemorrhages in the brain, occasional periportal and parenchymal mononuclear cell infiltrates in the liver, and hemosiderin-containing macrophages in the spleen. No significant lesions were found in the lungs, heart, or kidney. The AHL pathologist then requested blood samples of herd mates, which were collected randomly from the tail vein of 3 cows on day 11. These blood samples were collected into EDTA and clot activator vials and stored in the refrigerator until being sent to the AHL. The complete blood (cell) count (CBC) did not reveal any significant trend among the 3 cows. However, the biochemical profile consistently demonstrated moderate to markedly reduced serum magnesium levels, moderate to markedly reduced levels of urea, and 2 of the 3 samples revealed moderate to markedly reduced beta-hydroxybutyrate levels. The latter 2 changes indicate a chronic poor plane of nutrition or starvation. A diagnosis of hypomagnesemia caused by lack of appropriate mineral supplementation in a known magnesium deficient area was made. An increased awareness, generally promoting supplementation by the farmer, likely makes this condition currently uncommon in this area.

The owner was instructed to supplement his cows with magnesium oxide powder mixed with 2 parts salt, to be

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Table 1. Results of serum magnesium levels in cows from a cow/calf operation in Ontario that experienced mortality due to hypomagnesemia. Tests performed by the Animal Health Laboratory, Guelph, Ontario, on whole blood collected from the tail vein of cows, stored in EDTA tubes, and refrigerated until processing (RR = 0.82 to 1.30 mmol/L)

Cow	Sample 1 (day 11 after case presentation) mmol/L	Sample 2 (day 25, 12 d after commencement of supplementation) mmol/L
Black and White	0.40	0.80
Mousy	0.70	0.80
Blonde	0.60	—
Red and White	—	0.80

offered free choice. He was also notified that the blood magnesium levels would be rechecked in 2 wk. To confirm low levels of magnesium in the feed, samples of the first-cut hay that the herd was fed over winter, as well as of the fresh pasture grass, were collected on day 16. The samples were frozen and submitted for micronutrient analysis at Agri-Food Laboratories, Guelph, Ontario. The results revealed low levels of magnesium in the grass hay, 0.08% on dry basis (reference range [RR] = 0.11% to 0.23%). The fresh cut grass contained low normal levels at 0.12% magnesium dry basis (RR = 0.11% to 0.23%), and elevated levels of potassium at 2.76% (RR = 1.32% to 2.42%). Since the fresh grass was collected approximately 2 wk after the onset of the clinical signs, it is hypothesized that the levels of magnesium in the grass were, in fact, lower earlier in the growing season. A history of sudden death in cattle grazing on grass growing under cool, wet spring conditions is often diagnostic of hypomagnesemia (1–3).

The serum magnesium levels were rechecked on day 25, 12 d after magnesium supplementation commenced. Blood was collected from 2 of the same cows, and a third cow chosen at random (Table 1). These samples were placed in a vial containing no anticoagulant and refrigerated until being sent to AHL. The results seemed to demonstrate an improvement in the serum magnesium levels, though they were still low. The owner has been instructed to continue mineral supplementation. No more losses attributable to this condition have been reported on this farm.

Hypomagnesemia results from a primary dietary deficiency of magnesium. This condition has been seen in goats, sheep, and beef and dairy cattle (1–4), as well as in reindeer (5). It is a long-known disease of ruminants, but this case typifies winter tetany in beef cattle, a latent form of hypomagnesemia characterized by chronic energy deficiency and undersupplementation of magnesium. The etiology of this condition differs slightly from that of classic grass tetany (hypomagnesemia) in that clinical signs are triggered by a stressor, such as cold spring weather, but these cattle have actually had low levels of serum magnesium for an extended period of time (6). Magnesium is as an essential mineral in the activation of enzymes such as ATPases, kinases, and phosphatases; in RNA, DNA, and protein synthesis; and as a regulator of membranes and modulator of synaptic transmission in skeletal muscle (7–9). Thus, hypomagnesemic cattle may exhibit decreased productivity, teeth grinding, salivation, ataxia, recumbency, tetany, seizures, and death (1–4). The development of any form of hypomagnesemia is dependent on a balance of magnesium intake versus loss, which is determined by both host and environmental influences.

Host factors, such as decreased feed intake, a magnesium deficient diet, lactation, and altered absorption, lead to the depletion of intracellular magnesium levels and, eventually, serum magnesium (4,7,10). Magnesium is primarily absorbed in the forestomachs of ruminants. Rumen epithelium takes up magnesium by both paracellular (passive) and transcellular (active) mechanisms (4,8). Unlike the metabolism of many other essential minerals, there is no specific hormonal system to regulate magnesium homeostasis (1–4,8,10). The kidney is able to excrete excess magnesium, but it is unable to conserve enough magnesium systemically in situations of deficiency. As a result, output due to endogenous losses and exogenous losses, especially secretion in milk, exceeds the intake of magnesium, and the extracellular concentration of magnesium declines, while the losses continue.

When cattle are suddenly changed from winter rations to spring grass, decreased magnesium absorption may occur. Current literature is equivocal as to the importance of magnesium concentration in the grass itself as a factor leading to grass tetany. Some authors claim that it is a prerequisite (1–3), while others believe that this condition may occur under conditions of normal magnesium content (4). Fast-growing spring grass has been found to be high in potassium and crude protein, and low in sodium. These factors have a strong influence on magnesium absorption by the rumen epithelium. An increased intake of oral potassium results in a decrease in magnesium absorption from the forestomachs due to a decrease in the passive driving force of magnesium uptake by the epithelium (1–4,7–10). The decreased level of sodium and high levels of potassium in young spring grass leads to sodium deficiency, a decreased Na:K ratio in the rumen, and consequently an increased level of potassium in ruminal fluid, which further exacerbates the imbalance by decreasing the absorption of magnesium from the rumen (4). Lush grass innately has an increased level of crude protein. This factor, combined with increased use of nitrogenous fertilizers in the soil, causes an increase in ammonia in ruminal fluid, leading to a decrease in the availability and absorption of magnesium (4,8,9). Adequate amounts of fermentable carbohydrates are important in maintaining serum magnesium levels, since magnesium solubility and the absorptive surface area of rumen papilla both improve with availability of short chain fatty acids and lowered rumen pH (4). Stress may lead to clinical hypomagesemia, since sympathetic nervous system activation causes an epinephrine release resulting in a decreased plasma magnesium concentration (4,5).

Magnesium status of an animal is most conveniently determined by serum magnesium levels. It characterizes the state of magnesium metabolism in the animal, thus it is a good basic indicator of both dietary adequacy and nutritional status (10). It should be noted that mild low

serum magnesium levels may not predict clinical signs, since the neurological manifestations are reflective of cerebrospinal fluid (CSF) magnesium concentrations. The concentration of magnesium in the CSF is maintained over a wide range of serum magnesium levels. However, CSF concentrations of magnesium are related to serum in a linear pattern at markedly low levels of magnesium in the blood (4).

Prevention of hypomagnesemia should be aimed at 2 major goals: continuous provision of adequate levels of magnesium in the diet and maximizing absorption of this essential mineral. Possible choices of supplemental salts include magnesium carbonate, magnesium sulfate, and magnesium chloride, but magnesium oxide (MgO) tends to be the most common choice, based on economics, palatability, and fewer laxative effects than the other options (7,8). Improving absorption of magnesium by the ruminal epithelium can be attempted by lowering dietary potassium intake, though this may be impractical. Increasing magnesium intake by supplementing with MgO, offering salt lick blocks to prevent sodium deficiency, and increasing total energy intake through the provision of adequate amounts of fermentable carbohydrates are all effective tools in preventing the risk factors for developing grass tetany (4). These measures are especially important when moving from normal winter rations to a young spring grass pasture, and in lactating cows.

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